Pregnancy and fasting during Ramadan

SIR,—Awad H Rashed[1] and Jane Reeves[2] have discussed fasting by pregnant women during Ramadan. I worked in the Republic of Yemen for over 10 years on various projects in both inner city slums and remote rural areas. Pregnant women often fasted during Ramadan even though they did strenuous physical activity, such as collecting water or working in the fields.

In my experience lack of knowledge about exemption from fasting during Ramadan was not the most important issue. The main problem was that the women did not want to have to make the time up later, when they would be the only member of the household fasting. They preferred to fast in Ramadan, even though it made them tired and in some cases weak, because they wanted to participate fully in the religious festival. They often expressed their dislike of having to fast when no one else in the family was fasting but were adamant in their belief that they must fast for the full four weeks. I even know of a few non-pregnant women who took the contraceptive pill for the complete month of Ramadan, without the customary seven day break, so that they would not have to stop fasting because they were menstruating.

I would be interested to know if explaining the current teaching on fasting in pregnancy to Muslim women has any effect on their practice; I remain unconvinced. I would also be interested to know what are the effects of fasting on pregnant women. In Yemen for many women the diet improved in variety and quality, especially in the poorer households, during Ramadan. The main problems cited by the women were the restriction of fluid intake during the day and, especially in the last week of Ramadan, lack of sleep.

I agree with Rashed that more thorough scientific attention should be paid to this subject to ensure that we give Muslim women sound, useful advice in accordance with their religion.

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1 Rashed AH. The fast of Ramadan. *BMJ* 1992;**304**:521-2. (29 February.)
2 Reeves J. Pregnancy and fasting during Ramadan. *BMJ* 1992;**304**:843-4. (28 March.)

Cardiac stress during transurethral prostatectomy

SIR,—Julian W H Evans and colleagues report the haemodynamic effects of transurethral resection of the prostate.[1] Their earlier study of the subject stimulated us to look at it ourselves.[2]

We compared the haemodynamic changes in 22 patients undergoing transurethral resection of the prostate under general or spinal anaesthesia. The patients given general anaesthesia received a standardised anaesthetic regimen. Variables recorded included heart rate, arterial blood pressure, and ascending aortic blood flow assessed with Doppler aortovelography (Sci Med). This technique, which is analogous to the Doppler technique used by Evans and colleagues, accurately reflects trends in cardiac output as determined by other recognised techniques.[3]

In our patients given general anaesthesia the estimated cardiac output fell by a mean of 30% with induction of anaesthesia but returned towards the baseline value before surgical resection started. Once resection began the cardiac output fell by 5-10%, returning towards preinduction values after 40 minutes. At the beginning of resection mean arterial pressure was 10% below preinduction values, and it remained at this level throughout resection. The heart rate before the start of resection was 25% below preinduction values and gradually returned to baseline values after 40 minutes of resection. Calculated systemic vascular resistance increased by 40% with induction of anaesthesia but returned to baseline values before resection started and remained stable thereafter.

Haemodynamic variables in the patients given spinal anaesthesia were stable throughout the resection.

Thus the only appreciable haemodynamic changes that we observed were related to induction of anaesthesia and not to prostatic resection. In our hospital irrigating fluid is warmed to body temperature, and we believe that the changes that Evans and colleagues found may simply reflect the effect of irrigating the bladder with large volumes (>11 litres per patient) of cool fluid.

Evans and colleagues conclude that cardiac stress was increased in their patients through an increase in left ventricular afterload. Left ventricular afterload is determined by left ventricular end diastolic diameter and systemic arterial pressure.[4] Alterations in systemic vascular resistance (derived from cardiac output and blood pressure gradient) will affect left ventricular afterload only if they result in a change in arterial pressure. The only estimate of afterload in Evans and colleagues' study was the mean arterial pressure, which did not increase significantly. Also, other important determinants of cardiac work—that is, heart rate and stroke distance—both fell significantly, thus tending to reduce cardiac work. Therefore, we believe that the conclusion is unsubstantiated.

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1 Evans JWH, Singer M, Chapple CR, Macartney N, Walker JM, Milroy EJG. Haemodynamic evidence for cardiac stress during transurethral prostatectomy. *BMJ* 1992;**304**:666-71. (14 March.)
2 Evans JWH, Singer M, Chapple CR, Macartney N, Coppinger SWV, Milroy EJH. Haemodynamic evidence for peroperative cardiac stress during transurethral prostatectomy: a preliminary communication. *Br J Urol* 1991;**67**:376-80.
3 Schuster AH, Nanda NC. Doppler echocardiographic measurement of cardiac output: comparison with a non-golden standard. *Am J Cardiol* 1984;**53**:257-9.
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SIR,—Julian W H Evans and colleagues noted large increases in mean arterial pressure and systemic vascular resistance within two minutes of starting prostatic resection compared with values in controls undergoing herniorrhaphy.[1] These increases, they postulate, are secondary to the release of a vasoactive compound from the prostate.

Baseline blood pressure and aortic blood flow (oesophageal Doppler ultrasonography) were measured, and systemic vascular resistance and cardiac output calculated, after induction and before surgery, but the group having prostatectomy had been put into the Lloyd-Davies position. This would considerably increase venous return, with a compensatory inhibition of vasoconstrictor autonomic outflow via atrial and pulmonary volume receptors and baroreceptors. The study group would therefore have a lower baseline systemic vascular resistance compared with the controls and a variable baseline cardiac output depending on the response to the autotransfusion. If a vasoconstrictor stimulus is applied to both groups it may have a more profound effect on the study than the control group; certainly the two groups would not be comparable.

The authors then exclude a pressor response to surgery by saying that no lacrimation, sweating, or tachycardia was seen. These are, however, relatively coarse signs seen in response to somatic afferent stimulation. Autonomic afferent activity is involved in hormonal responses to surgery[2] and is not blocked without an extensive extradural technique. So a degree of vasoconstriction may occur secondary to released catecholamines, as well as in response to autonomic efferent activity, without much lacrimation or sweating. The authors suggest that these haemodynamic changes predispose to cardiac stress and difficulty tolerating irrigation loads and hypothermia.

If indeed these changes are related to increases in venous return perhaps invasive monitoring of central venous pressure should be considered more often, or even measurement of pulmonary artery pressure and cardiac output, as this would give more information than ultrasonography alone. A similar study with patients undergoing prostatectomy under extradural anaesthesia would be interesting.

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1 Evans JWH, Singer M, Chapple CR, Macartney N, Walker JM, Milroy EJG. Haemodynamic evidence for cardiac stress during transurethral prostatectomy. *BMJ* 1992;**304**:666-71. (14 March.)
2 Analgesia and the metabolic response to surgery [editorial]. *Lancet* 1985;i:1018-9.

AUTHORS' REPLY,—We are pleased that P M S Dobson and colleagues agree with our hypothesis that cold stress could be an important factor in causing perioperative circulatory disturbances. Indeed, we are about to submit for publication a report of a further study confirming this concept.

The changes seen in our patients having transurethral prostatectomy could be explained by a primary increase in afterload with a secondary fall in cardiac output or by primary myocardial dysfunction with compensatory vasoconstriction. As no changes were seen in the control group, who received a similar anaesthetic and had stable core temperatures, we suggested that rapid central cooling causing vasoconstriction together with increases in whole blood viscosity was the primary event.

In response to S N Gower, the legs are raised only minimally in the Lloyd-Davies position compared with the lithotomy position. Therefore increases in venous return are unlikely to be important. We did not suggest that the haemodynamic responses predispose to cardiac stress, but that they are a reflection of it.

We cannot agree with Gower's recommendation for more frequent monitoring of central venous pressure as changes in ventricular compliance and peripheral vascular tone will disrupt any relation between right ventricular end diastolic pressure and volume. Pulmonary artery catheterisation should not be regarded as a routine procedure in these patients in view of its morbidity, cost, and logistical implications.

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Psychological treatments in cancer patients

SIR,—Steven Greer and colleagues present further compelling evidence to suggest that a large proportion of patients diagnosed as having cancer suffer from psychological problems and that these problems are eminently treatable with psychological therapy.[1] Given the overwhelming